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Health and Environmental Effects Profile for Butylate

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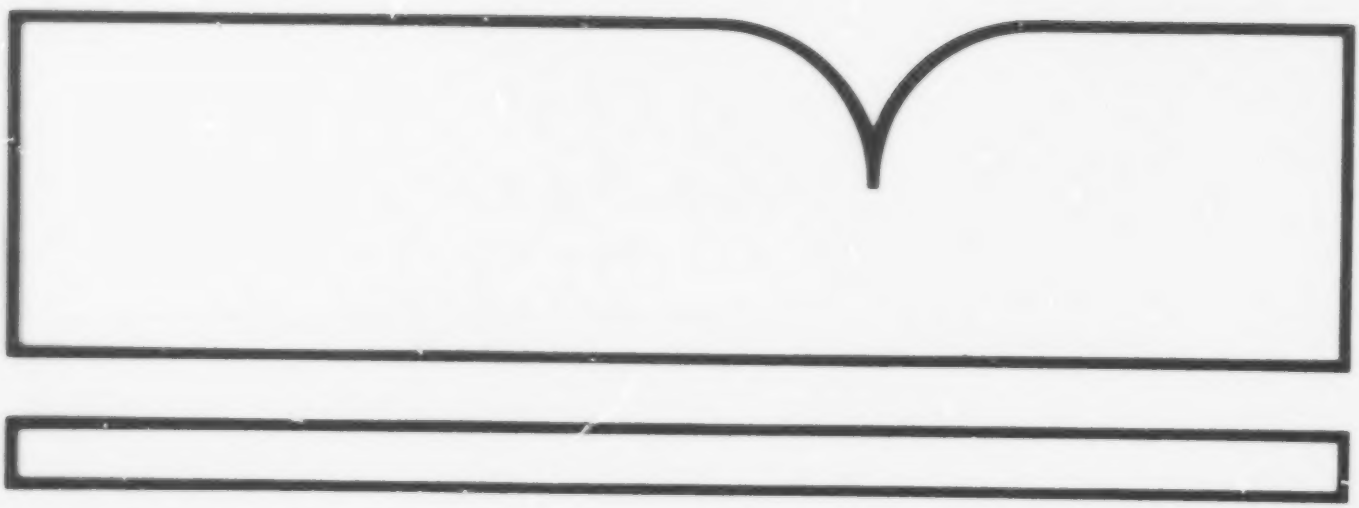

U.S. Department of Commerce
National Technical Information Service
NTIS

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January 1984

HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR BUTYLATE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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16. ABSTRACT

The Health and Environmental Effects Profile for butylate was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Butylate has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for butylate is 1×10^{-1} (mg/kg bw/day) for oral exposure.

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

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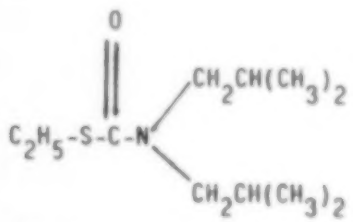
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
GC	Gas chromatography
GSH	Glutathione
i.p.	Intraperitoneal
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
ppb	Parts per billion
ppm	Parts per million
TLC	Thin layer chromatography

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Butylate is the common name for S-ethyl di-isobutylthiocarbamate (BCPC, 1977). It is also known as carbamothioic acid, bis(2-methylpropyl)-, S-ethyl ester, S-ethyl bis-(2-methylpropyl)carbamothioate, ethyl N,N-diisobutyl thiocarbamate, S-ethyl-diisobutyl thiocarbamate and ethyl-N,N-diisobutyl thiolcarbamate (NIOSH, 1983). This compound was introduced into the market in 1954 under the code number "R-1910" and has the trade name Sutan. The structure of butylate is depicted below:



The molecular formula of butylate is $C_{11}H_{23}NOS$, and the molecular weight is 217.41. The Chemical Abstract Service (CAS) registry number is 2008-41-5.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Butylate is a clear liquid with an aromatic odor (BCPC, 1977). Some of the physical properties are listed below:

Boiling point:	71°C at 10 mm Hg (Ouellette and King, 1977)
	138°C at 21 mm Hg (BCPC, 1977)
Vapor pressure:	1.3×10^{-3} mm Hg at 25°C (BCPC, 1977)
Density (temperature unspecified):	0.9417 (BCPC, 1977)
Solubility in water:	45 mg/ℓ at 20°C (BCPC, 1977; Ouellette and King, 1977)
Sorption coefficient (K_{oc}) (calculated from water solubility):	540 (Kenaga, 1980)

Butylate is miscible with kerosene, isobutyl methyl ketone and xylene (BCPC, 1977).

1.3. PRODUCTION DATA

Butylate is formed from a 2-step reaction in the first step di-isobutyl-amine is reacted with posgene to produce carbamoyl chloride. Carbamoyl chloride is then reacted with ethanethiol to produce butylate (BCPC, 1977). It is produced by the Stauffer Chemical Company in Cold Creek, AL, and St. Gabriel, LA (SRI, 1983; Ouellette and King, 1977). The U.S. production data for this compound were unavailable (see Appendix).

1.4. USE DATA

Butylate is used as a preplant herbicide to control broad-leaved and grass weeds in corn. It is toxic to germinating seeds and is mixed with the soil prior to planting (BCPC, 1977; Ouellette and King, 1977).

2. ENVIRONMENTAL FATE AND TRANSPORT

The fate and transport of butylate in the soil due to microbial degradation has been studied extensively. While fate and transport also are likely to depend on chemical or photochemical reactions, no information is available on such reactions in the sources cited in the Appendix.

2.1. SOIL

Microbial degradation can be an important factor in reducing the persistence of butylate in soil under conditions where volatility is reduced and environmental factors are favorable (e.g., moderate organic matter, increased aeration, and increased moisture or temperatures) (Kaufman, 1967). Microorganisms capable of the degradation of this compound have not been identified; however, the probable degradation pathway has been described as involving hydrolysis at the ester linkage, with subsequent formation of a mercaptan, secondary amine and CO_2. The mercaptan could then be converted to an alcohol by transthiolation and further oxidized to an acid prior to entering the metabolic pool (Kaufman, 1967).

The degree of degradation of butylate in the soil depends on the nature of the soil. For example, in soils with a moderate adsorptive capacity, herbicides are significantly degradated by microorganisms, whereas in soils with a low adsorptive capacity, volatilization may be the primary route of dissipation (Koren et al., 1968). Preliminary results obtained by Kaufman (1967) also suggest some correlation between chemical structure and soil persistence of thiocarbamate herbicides. Thiocarbamates having branched-chain alkyl groups were more persistent in soil than those having similar straight chain alkyl groups. The persistence of butylate in a silty loam soil was ~100 days as compared to 60 days for pebulate and ethyl N,N-dipropylthiocarbamate.

Transport of butylate in soil may be controlled by volatilization and leaching. The stronger the adsorption of a herbicide to soil particles, the less likely its chances for volatilization. Koren et al. (1969) found that the degree of adsorption of a herbicide to soil is directly correlated with the content of organic matter of the soil. Weidner (174) studied the adsorption and depth of leaching of butylate on three different soil types with organic matter and clay contents ranging from 0.07-4.55 and 5-33%, respectively.

Adsorption increased as the content of organic matter and clay increased, and leaching increased as the contents of these two constituents decreased. Similar results were obtained by Elker et al. (1981), who found that adsorption of butylate was highest on peat and lowest on sand.

2.2. AIR

Although no data directly addressing the fate and transport of butylate in the atmosphere were encountered, some inferences can be made. The two likely routes of entry into the atmosphere are spray drift during application and volatilization from soil and water. Vaporization will occur more readily from soils with low clay and organic matter and higher moisture content. Studies by Koren et al. (1969) found that regardless of the soil type, a relationship existed between the volatility and the vapor pressure of a herbicide; however, no specific data were available for butylate.

The fate of butylate in the atmosphere will depend upon its reactions with O_3, NO_x and free radicals (e.g., RO•, O• and RO_2•) and its ability to undergo photolysis; however, no information was encountered that described the significance of these reactions. Butylate may be found in the atmosphere in the particulate-sorbed phase based on the calculated sorption

coefficient of 540 (Kenaga, 1980). Considering the water solubility of this compound (45 mg/ℓ) it is possible that the particulate-sorbed compound may be removed from the atmosphere by both wet and dry deposition.

2.3. WATER

Butylate can enter the aquatic media through runoff and leaching from soil. While no studies relating to the fate or transport of this compound in aqueous media were found among the sources in the Appendix it could be determined by the usual range of processes, hydrolysis, photodegradation, free radical reactions and microbial degradation. Studied in soil suggested that biodegradation may be a significant process for removal of butylate from water. However, based on the reported sorption coefficient and the vapor pressure of 1.3×10^{-3} at 25°C, butylate is more likely to be transported via adsorption onto suspended solids.

3. EXPOSURE

STORET contained 76 observations for butylate in water ranging from 0.0-4.7 µg/ℓ, with a mean concentration of 0.7 µg/ℓ. The data for the 13 stations sampled are summarized in Table 3-1.

One additional study identified butylate in well water in Ontario, Canada, at concentrations of 0.1-1.0 µg/ℓ (Frank et al., 1979).

TABLE 3-1

Monitoring Data for Butylate*

Station/Description (ambient/stream)	Range (µg/ℓ)	Mean (µg/ℓ)
Missouri River, Grand Chariton	0.8-2.0	0.909
Missouri River, Grand Chariton	0.8-4.7	1.45
Missouri River, Grand Chariton, ambient lake	0.8-4.2	1.48
Missouri River, Grand Chariton	0.8-4.7	1.45
Missouri River, Grand Chariton	0.8-2.0	0.933
Cedar River Basin, Cold Water Creek	0.44	0.44
Cedar River Basin, Spring Creek	0.0-0.0	0.0
Soldier River Basin, East Soldier Creek	0.2-0.8	0.5
Big Sioux River Basin	0.02-0.14	0.062
Chariton River Basin	0.1-0.1	0.1
Chariton River Basin, S. Fork Chariton River	2.3	2.3
Soldier River Basin, Emigrant Creek	0.3	0.3
Chariton River Basin, Lake Rathbun	0.8-4.2	1.48

*Source: STORET

4. PHARMACOKINETICS

4.1. ABSORPTION

Data relating specifically to the absorption of butylate were not located in the available literature; however, some information was obtained from a metabolism study by Hubbell and Casida (1977). Male albino Sprague-Dawley rats, 190-210 g, were administered by gavage 12.3 or 156.0 mg/kg radiolabeled ^{14}CO-butylate. Within 48 hours, 31.5 and 27.3% of the administered radioactivity was recovered in the urine and 60.9 and 64.0% was expired as $^{14}CO_2$. These results indicate that butylate is absorbed from the gastrointestinal tract of rats.

4.2. DISTRIBUTION

Hubbell and Casida (1977) measured the tissue radioactivity 48 hours after the administration of 12.3 and 156.0 mg/kg ^{14}CO-butylate to rats by gavage. At the low dose, 2.4% of the administered radioactivity was retained in the body with levels of radioactivity equivalent to 276 ppb in the blood, 524 ppb in the kidney, 710 ppb in the liver and a range of 182-545 ppb in other tissues (brain, fat, heart, lung, muscle, spleen and testes). At the high dose, 2.2% of the radioactivity was retained in the body with 2076 ppb in the blood, 5320 ppb in the kidney, 7720 ppb in the liver and 1720-5560 ppb in other tissues.

4.3. METABOLISM

Casida et al. (1975) studied the sulfoxidation of butylate by mouse liver microsomes in vitro. When ^{14}C-labeled preparations of butylate were incubated with mouse liver microsomes, butylate sulfoxide was detected as the major metabolite. Butylate sulfone was not among the metabolites detected, and one minor ninhydrin-positive metabolite was found. The extent of butylate metabolism increased when soluble enzymes and reduced glutathione (GSH) were added to the incubation system. A volatile mercaptan was

liberated, and the sulfoxide recovery decreased. These findings suggested that reaction of GSH with butylate sulfoxide results in a cleavage of the carbamoyl bond by GSH attack at the carbonyl group (perhaps catalyzed by GSH-S-transferase), liberating either the mercaptan or sulfonic acid (Casida et al., 1974).

Hubbell and Casida (1977) further characterized the metabolism of butylate based upon identification of the 48-hour urinary metabolites of ^{14}CO-labeled preparations of butylate (12.3 or 156 mg/kg) or butylate sulfoxide (6.15 mg/kg) administered to rats by gavage, or S-(N,N-diisobutylcarbamoyl)-glutathione (0.08 mg/kg) or S-(N,N-diisobutylcarbamoyl)-cysteine (0.13 mg/kg), administered by i.p. injection. Approximately 40% of the administered ^{14}CO-butylate was metabolized by ester cleavage and $^{14}CO_2$ liberation without going through the sulfoxide as an intermediate. The metabolites from all compounds were essentially the same qualitatively and quantitatively. The metabolites for ^{14}CO-butylate included, as percent of urinary radioactivity, 4.3% as the N,N-diisobutyl mercapturic acid, 17.1% as the N-isobutylmercapturic acid, 0.8% as the mercaptoacetic acid derivative, 11.7% as the glycine conjugate of the mercaptoacetic acid derivative and ~66% as at least 15 other metabolites. In addition, the mercapturic acid was detected by GC as 4-6% on a molar basis of the administered dose in the urine 24 hours after i.p. injection of 1.0 mmole butylate/kg to rats, and the GSH conjugate was detected by TLC in rat livers 30, 60 and 120 minutes after i.p. injection of 1 mmole/kg butylate sulfoxide.

4.4. EXCRETION

Hubbell and Casida (1977) administered 12.3 and 1560 mg/kg of $^{14}CO_2$-butylate by gavage to 190-210 g male rats. Within 24 hours, 60.9 and 64.0% of the administered radioactivity was expired as CO_2, 27.3 and 31.5% was excreted in the urine and 3.3 and 4.7% in the feces, respectively.

5. EFFECTS

5.1. CARCINOGENICITY

No information was encountered in the available literature on the carcinogenicity of butylate, and this compound is not scheduled for testing by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

Butylate failed to induce reverse mutations in the spot test in any of five strains of Salmonella typhimurium (TA1535, TA1537, TA1538, TA98 and TA100) with and without the S-9 activating fraction (Eisenbeis et al., 1981). Butylate failed to increase the rate of dominant lethals in Drosophila melanogaster, but increased the frequency of sex-linked recessive lethals (Murnik, 1976). NIOSH (1983) classified butylate as a mutagen based on a Russian study by Pilinskaya et al. (1980) who evaluated the potential mutagenic hazards of 22 pesticides from their cytogenic activity in mice. No other information on this study was available from the abstract.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenic potential of this compound were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding reproductive effects of butylate were not located in the available literature.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the toxicity of this butylate following chronic exposure were not located in the available literature.

5.6. OTHER RELEVANT INFORMATION

Limited information on the toxicity of butylate was located. An oral LD_{50} value of 4000 mg/kg in rats was reported (NIOSH, 1983), a value typical for carbamate herbicides (Murphy, 1975).

6. AQUATIC TOXICITY

6.1. ACUTE EFFECTS

Acute toxicity tests of butylate have been conducted with one species of freshwater invertebrate and three species of marine animals. Sanders (1970) reported that the 96-hour LC_{50} of two butylate formulations for freshwater amphipods, Gammarus fasciatus, were 10 and 15 mg/ℓ. Johnson and Finley (1980) reported a 96-hour LC_{50} of 11 mg/ℓ for the same species. Butler (1965) reported that exposure to 1 mg/ℓ had no effects on marine shrimp, Penaeus aztecus, and spot, Leiostomus xanthurus, during 48 hours of exposure and no effects on shell growth of oysters, Crassostrea virginica, during a 96-hour exposure period.

6.2. CHRONIC EFFECTS

Pertinent data regarding the chronic effects of butylate on aquatic animals were not located in the available literature.

6.3. PLANT EFFECTS

The only information concerning the effects of butylate on aquatic plants was provided by Hess (1980), who reported that the growth of algae, Chlamydomonas eugametos, was inhibited 83% by 48 hours of exposure to 22 mg/ℓ, but was not inhibited by 2 mg/ℓ.

6.4. RESIDUES

Pertinent data regarding residues of butylate in aquatic organisms were not located in the available literature. However, on the basis of water solubility, Kenaga (1980) calculated a predicted BCF of 72, indicating limited potential for bioaccumulation.

6.5. OTHER RELEVANT INFORMATION

Other relevant information concerning the effects of butylate on aquatic organisms was not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Tolerances were established at 0.1 ppm under the Federal Food, Drug and Cosmetic Act for negligible residues of butylate in or on corn grain, fresh corn and corn forage and fodder (33 FR 2844-5).

7.2. AQUATIC

Guidelines or standards for the protection of aquatic organisms from the toxic effects of butylate were not located in the available literature.

8. RISK ASSESSMENT

No data on the carcinogenicity of butylate were located in the available literature. Negative results were obtained in *in vitro* mutagenicity assays with bacteria both in the presence and absence of exogenous metabolic activation (Eisenbeis et al., 1981). Butylate failed to increase the rate of dominant lethals in *Drosphila melanogaster*, but increased the frequency of sex-linked recessive lethals, evidence of the production of point mutations (Murnik, 1976). Hence, there is limited evidence of mutagenicity for this compound. No data concerning the potential teratogenicity of butylate were located.

No information on the effects of chronic or subchronic exposure to butylate was located in the available literature; therefore, no ADI value could be derived.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

The CBI Appendix to this document reviews other animal data from which an ADI of 7 mg/day for a 70 kg man (0.1 mg/kg bw/day) was derived. The ADI was based on a NOEL of 10 mg/kg/day for decreased prothrombin time in rats in a chronic feeding study, and an uncertainty factor of 100 (10 to account for interspecies extrapolation and 10 for protection of unusually sensitive individuals within the population).

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

Most of these searches were conducted in February, 1983; a few were conducted March-May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. TLVs: Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981). Cincinnati, OH. p. 486.

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